UNITED STATES DISTRICT COURT
DISTRICT OF MASSACHUSETTS

*SILAS CALHOUN and EMILY CALHOUN, Individually and as Parents and Next Friends of ESTELLA CALHOUN Plaintiffs vs. *UNITED STATES OF AMERICA Defendant	*	CIVIL ACTION No. 04-10480-RGS

BEFORE THE HONORABLE RICHARD G. STEARNS
UNITED STATES DISTRICT JUDGE
CIVIL BENCH TRIAL, DAY 3
April 11, 2007

APPEARANCES:

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A I'm board certified in pediatrics, and I'm board certified in psychiatry and neurology with a special competence in child neurology.

Q Do you have other kinds of certifications?

A American Society of Neurorehabilitation, and probably that's it.

I don't know. You have my CV. I think that's it.

Q Do you have any current academic appointments?

A Yes, at Mass. General Hospital. And where I am at North Shore Children's Hospital is part of the Partners amalgam of hospitals. So we have residents from Mass. General coming through. So I do a teaching conference at Mass. General every week.

Q And with regard to that, have you received any awards at Mass. General?

A Mass. General, I was the Teacher of the Year Award in pediatric neurology twice in the last five years. I don't remember the exact dates.

Q How long have you been practicing pediatric neurology?

A Since 1971 with pediatric -- depending on how you look at it. I am a physician as of 1963, pediatric neurology starting with the residency, I guess, would be '68, but certified, somewhere '71, '72, I think, practicing full time since 1971.

Q Have you published any literature in your field?

a lot of testimony about the medical history here, and I want to focus in on particular issues with respect to some of the issues involved here. And first I would like to ask you -- you're familiar with Estella's early history and that she was admitted to Emerson Hospital suffering from severe hypernatremic dehydration?

A Yes.

Q Would you explain for the Court the effects of severe hypernatremic dehydration on an infant's brain?

A Sure.

Hypernatremia means a high sodium. Sodium normally would be in the blood, extracellular fluid, up to 146, I believe, and she was 172. So she had severe hypernatremia.

What happens is the -- there's a drive towards equalizing the osmotic content from inside the cells and into the extracellular fluid. And since there's a lack -- in another words, in order to equalize that or lower the serum sodium, the cells, the nerve cells, or all the cells actually, excrete water into the extracellular space so as to lower the serum sodium. In so doing, the cell itself shrinks, all the cells that is, shrink, and then a number of known and unknown processes take place.

The cells manufacture molecules in an attempt to equalize the fluid, keep the fluid from leaving the cells and preserve the structure of the cells. So those molecules

are manufactured, and the attempt then is to keep the fluid from continuing to excrete into the extracellular space.

So the main mechanism is within each cell that takes place, some known, some unknown, that also -- not only do you have the shrinkage of the neurons of the nerve cells, but you have disruption of their processes, their metabolic processes, again some known factors and some unknown. So it's a diffuse process that takes place throughout the brain.

What happens in a grosser sense is that the fluid leaving the cells make the brain shrink. When the brain shrinks, it can pull away from the coverings of the brain, arachnoid and dura, and those coverings have blood vessels that run in and out of the brain, and sometimes when the brain shrinks those vessels are torn. So you get bleeding or hemorrhage.

You also have a formation of clots or thrombi in the veins, particularly within areas -- many areas of the brain, and the clots or the thrombi prevent the flow of blood that originally comes from the arteries and goes back through the brain and then out the veins or the sinuses. You see the sagittal sinus is one the big veins, for example.

So when there is this clotting of the blood in the veins, then the pumping in of blood from the other side, the

arterial side, makes the brain congested. So you get further disruption of cellular functions.

So there's a diffuse process, some of which can be seen grossly with clotting in the veins and tearing of blood vessels, and some of which is on a microscopic or submicroscopic level throughout the brain.

That's pretty much what -- at least my understanding of what happens with hypernatremia.

And an infant's brain is presumably more susceptible to these injuries than an adult's. Although I'm not 100 per sure that's so either, but the adult brain can sustain a little bit more of this than an infant's brain can.

Q Doctor, I'd like to go over portions of the record at Children's Hospital with you, and perhaps we can start with the emergency department record, and there is a flow sheet, if you turn to page 150, 0150.

A I have it.

Q It looks like on the left here there is a time given starting at 2100.

A I see that.

Q And then as we go across it talks about the observations made?

A Yes.

Q So I see at 2115 -- could you explain what that is?

A So there is a one minute focal seizure of the left arm and face, and they gave Ativan, which is a Valium-like drug that's used to stop seizures.

And so with the seizure of the left arm, face and leg, it says there was a color change and there are cries, multiple episodes of apnea, which is cessation of breathing, and seizures.

So apparently over the course of -- it looks like the next one was 2130, 15 minutes. Yes. There's 2120 and 2130.

Over that course of that 15 minutes, there were multiple episodes of seizure activity and cessation of breathing.

Do you want me to go on?

Q Yes, please.

A Intermittent left-sided seizures, intermittent apnea. The apnea resolved with simulation. Then they gave atropine, midazolam and succinylcholine. They were intubating probably. 3.5, that's the size on the endotracheal tube. So they intubated the baby and placed her on a respirator to sustain respiratory efforts, because they were continuing to give Ativan and then Phenobarbital, both of which can suppress respiration, in addition to whatever the seizures themselves were doing.

So that was from 2115 to 2150. So that's three

quarters of an hour, I guess, over that period of time.
Q Then if you turn to -- it continues now on page 152.
A Yes.
Q Starting on the left there it looks like 2155?
A Right.
So there is more Phenobarbital given and more Fentanyl. These are all drugs to paralyze the baby or to treat the seizure activity, Ativan.
The paralysis was because the baby was on an intubator -- was on a respirator, intubated. They didn't want the baby fighting the respirator.
Q I see at 2140 more Ativan was given and there were more seizures?
A Yes.
Then transferred to the ICU, I guess.
Q Now, I just want to direct you to the note of Dr. Andre du Plessis. That's No. 186.
A I have it, yes.
Q First of all, who is Dr. du Plessis?
A He's a pediatric neurologist at Children's with particular expertise in neonatal neurology.
Q I don't know if we need to go through the whole note here, but if you go down to the Impression section, which is near the bottom?
A Yes.

Q Could you read for the Court what that says.
A It says, neonatal encephalopathy with seizures of postnatal onset and associated with hypernatremic dehydration with possible -- looks like cerebral vein thrombosis and intracranial hemorrhage. I would check the MRI/MRV/MRA/EEG. Spinal tap for opening pressure, and I guess other studies, coagulopathy. See if there was an underlying -- well, what happens sometimes in a situation like this, you have what's called disseminated intravascular coagulation where there is -- processes take place within the blood to predispose towards more clotting.

They were looking for that or to see, I suppose, if there was an underlying abnormality of blood-clotting mechanisms that might have predisposed toward either the hemorrhage or the clots.

So those are the studies he was recommending.
Q Doctor, what do you understand the word "encephalopathy" to mean?
A Encephalopathy means brain dysfunction, and usually it's a diffuse term -- I mean it reflects diffuse dysfunction of brain processes.
Q Now I'd like you to turn to 166, which is Dr. du Plessis's note of the following day.

The baby, correct me if I'm wrong, the baby was admitted -- Estella was admitted -- on March 9. You just

thalamus?

A Well, it's the central way station, receives information from the cortex and other parts of the brain. It processes and sends out back to the cortex and to other areas in the brain. So it's sort of a central way station, processing information, maybe programming and organizing behaviors.

The brain stem is down below where the breathing and heart rate and so on originate.

I think that's probably enough said on that. I'll put it back together again for a minute.

So then -- and the veins run the -- there is a mention about the central sinus, I think, and that's -- it would be a large vein that runs along the middle of the brain.

So this is a diagram of the brain from underneath and on top and sideways and so on.

I think probably what's important to note is that within the substance of the brain are these bluish structures. They're like balloons. They're ventricles, and within these ventricles the cerebral spinal fluid is formed at a fixed rate all day long and the fluid flows in certain pathways back and forth to both sides of the brain, and then down through the middle and ultimately down around the spinal cord. So if someone has a spinal tap, that's the fluid that's in contiguity with the fluid that's up and

around, and it circulates over the surface of the brain and so on.

So these ventricles or spaces, balloon fluid-filled spaces, there's mention, I believe on the right side, the posterior horn of the right ventricle there was a bleed. So these are bleeds that are grossly seen when the brain shrinkage has torn blood vessels, veins primarily, in the region where the blood appears.

So that's a kind of localized finding from the brain shrinkage, but presumably there's diffuse abnormalities all over the brain because of the cellular defects -- cellular abnormalities that take place, metabolic abnormalities.

Also question about recurrent seizure and whether substances called excitotoxins are released and have further injuries -- some -- I mean, there are many processes that take place in the midst of seizures and what it leads to, what was encephalopathy, beyond just a bleed here or a bleed there. That's what you can see grossly.

But, anyway, that's the brain.

Q Let me get to that, and I'd like to revisit a couple of these issues.

A Sure.

Q It appears that on the 10th -- there was actually a CAT scan done on the 9th, then on the following day there was an

at microscopic slides or, to some extent on an x-ray or MRI.

The implication is that there is death of tissue enough to show up in whatever way you're looking, depending on how fine your magnifying glass is.

Q Is it known to you in your experience and to pediatric neurologists that damage can take place in the absence of an actual picture of that or -- the absence of an actual infarction that shows up on a film?

A Yes, I would think.

Certainly bleeding can damage nerve cells. Lack of oxygen can cause hypoxia, for example, or lack of oxygen, carbon monoxide poisoning, or apnea, whatever, that can cause damage, it's well known, to the brain. You may not see any infarction as such, which implies a blood vessel stopping circulation at that point.

I mean you could have many kinds of damage to the nerve functioning or central nervous system function without the actual physical finding of a death of tissue large enough to show up on whatever it is, CAT scan or MRI or pathology. You know, when you get to the ultrastructural level you can see it, but even then you may not see it. It's only functional. I mean, there's all levels of dysfunction, I suppose, some of which can be seen on an x-ray and some can't.

Q Now, there was another MRI done on the 14th. That's

And the important findings in this were that there were excessive right frontal and temporal sharp waves.

So in the right frontal region and the right temporal region there were abnormal electrical findings, I guess is what I would summarize it as. And it has potential in the sense that electrical -- it indicates a lowered electrical threshold for depolarization. And in that setting then the left-sided seizures, since the right side of the brain controls the left side of the body, electrical abnormalities in the right frontal region and temporal region would likely, if they were to evolve into a seizure, show themselves in abnormal movements on the left side of the body, which indeed is what we saw.

Q So again, Doctor, could you, for the Court, summarize the nature of the injury to the brain in summary terms and in general terms what that all describes?

A So as a result of abnormalities in the sodium content of the blood, various shifts in fluid and abnormalities in cellular processes took place.

On a gross sense, the brain shrunk because all the cells had their fluid leaving to try to equalize the osmotic gradient in the blood in the extracellular space. But in a gross sense the brain shrunk in different parts and various blood vessels were torn resulting in bleeds.

Along with that, there were clots formed within the

veins which adds to further congestion and abnormal metabolism within the brain. Some of that showed on an MRI. Some of that could be implied from the clinical findings of encephalopathy and the diffuse -- for example, abnormalities in the lower extremity reflexes, these all would point to other areas of the brain being involved.

And then the baby had seizures, which are abnormal electrical discharges with activation of the underlying brain, primarily from the right hemisphere, frontal presumably more than thalamus because the motor system is more frontal, although thalamus is involved in all back-and-forth sending of messages up and down the brain.

Q What was the treatment upon discharge?

A So on discharge the baby was continued on Phenobarbital as a seizure prophylaxis.

Q Now, again I'm not going -- we're not going to go through all the history of what happened for each provider, but I would like to direct you to Document No. 291.

A I have it.

Q And this is a note from Dr. du Plessis?

A Yes.

Q I wonder if you could just go down to the end of the first page.

A Yes.

Q And at the end of that paragraph where it says, "On

Q Yes.
A In summary, Estella's a three-year-old girl with a history of an extensive dural vein thrombosis, subsequent hemorrhagic infarction in the newborn period --
(Reporter interrupts.)
A I'm sorry. I'll do it much slower.
"In summary, Estella is a three-year-old girl with a history of an extensive dural vein thrombosis and subsequent hemorrhagic thalamic infarction in the newborn period. She has been doing rather well since that time with appropriate development. At this point she has several behavioral issues related to impulsivity, inattention, and hyperactivity that are often seen in children with this history of neurological injury."
Q Now, what is your understanding -- is that your experience as well?
A That -- my experience of --
Q With respect to -- with respect to those kinds of deficits and behavioral problems continuing to be seen even years after the --
A -- history of neurological injury?
Q Yes.
A Yes. Those are well-described aftereffects of neurological injury in children, newborns and older children.

impressions of people who are living with the child.

And so a dimensional or categorical approach would be you can have attentional problems and you may not fit all of the exact criteria for that particular diagnostic entity. I'm more in that category. But in actual fact, she fulfills enough criteria based on her parents' observations to say that her behavior fits into what we now call ADHD. And the question is, you know, are other people, the soccer coach, the after-school program, people who see her in different settings, is there a consensus? Is she different in different settings?

These are the kinds of things that one would want to have. I didn't have everything. I had some of that information, and based on my observations, my review of the medical records, my interview with the parents, I, to the more-likely-than-not, or whatever the characteristic phraseology is, that I felt as though she fulfilled criteria for attention deficit hyperactivity disorder.

And I was more concerned, or somewhat concerned -- I don't know about more concerned -- about what I thought might be clues to cognitive functioning in terms of organization, planning, inferential reasoning. You can't really tell that too well at the seven-year-old level. I don't know that I'm the best person to judge all that, but I have seen enough kids with this kind of behavior to worry

about planning and organizing, which is also frontal lobe dysfunction and may be more important than, you know, miles per minute activitywise, at least in the future anyway. The restlessness, the impatience that I saw was of more concern to me anyway.

Q So let me ask you --

A Sure

Q -- do you have an opinion to a reasonable degree of medical certainty as to whether the injuries that Estella suffered in this neonatal period were a substantial contributing factor in creating or causing the behaviors that you've noticed and others have noticed, as well as some of the cognitive problems that have been uncovered?

A Yes, I do.

Q And what's the opinion?

A My opinion is that the neurological insult that she suffered in the first two weeks of life have had a substantial -- are a substantial contributing factor to the current dysfunctions which have been enumerated previously, and that they are likely -- more likely than not causative or a significant contributing element to the causation of her current dysfunctions, which can, for want of a better term, be labeled ADHD.

Q I would like you to explain for the Court this entity ADHD. Could you give the Court some understanding of how we

In any event, not to go through too many digressions, but over time the labeling has changed and the characteristics have been more refined, or at least consensus has been achieved.

So it went from minimal brain damage to minimal brain dysfunction, which sounded a little less scary, and then minimal cerebral dysfunction, which sounded even less ominous.

And then parents took ahold in the '60's consumerism, and they said, you know, it isn't always brain injury. You can't always prove it. Why don't we just call it as we see it.

So then organizations for learning disabilities, organizations for attention deficits, were formed, and the labeling changed according to those responses to consumerism, really, is the way I look at it anyway.

In any event -- so there was no question over this history of 100 years that injury to the brain can result in behavioral differences in children.

There was a whole curriculum how to teach these children. Put them in a carol, reduce distractions.

What came about in the last 25 years is the understanding that maybe only a minority of children that present in this way have brain injuries that can be proven, and that maybe there is some other activity or factor that

evolved, seemed to be genetics. It seemed pretty well proven that genetics are responsible for the vast majority, not all, but the majority of children who present with these behavioral characteristics.

There is specialization and some research that suggests different circuits, frontal lobe circuits, are very popular because the frontal lobe does the things that I mentioned that it does that over time helps you to judge the consequences of your behavior, modulates your behavior, resists impulsivity and so on.

So what are the circuits involved? What are the chemicals involved? Are there differences in the genes that might explain this? And there's a lot of hypotheses, and there's no test at this point that somebody can send a blood test or an x-ray and say, yes, you've got it or not. It's a clinical diagnosis based on the kinds of things that I've talked about.

So that sort of brings us to where we are now. That does not negate the fact that even though the majority of children with these signs and symptoms may be on a genetic basis, that's not to take away the foundations of the observations that led to this whole understanding of the entity, which is that it can be an effect of brain injury.

And in my experience, which I have had a great deal of experience. I was in a rehab hospital for 20 years. I

mean I saw plenty brain injuries and the effects often are just that, even in the absence of a definite IQ difference you can see disinhibition and distractibility and impulsivity.

And as the children get older you worry about more significant things, not so much miles per minute and how much they run around, but what their patience will be like and what their ability will be to stick to tasks, form relationships, those are the issues that are worrisome to families in adolescence. And I'm giving a talk on that, so I'm sort of up on that subject, next fall at Harvard, a postgraduate course on pediatric neurology on the adolescent with this condition, because the issues are not so much jumping around as they are inner restlessness.

So, anyway, that's probably enough for one day.

Q You used the term "disinhibition."

A Yes.

Q In terms of its neurological basis in the brain --

A Well, the frontal lobes have a lot to do with inhibiting lower centers, and it's known, for example in head injuries, you often -- the brain -- there's acceleration, deceleration forces that take place in a head injury often with sliding of the base of the frontal lobe against this -- actually you can see it here (indicating).

The base of the skull here is rough, and where the

frontal lobes -- the bottom of the frontal lobes sit. So if you have a back-and-forth movement, you're going to injure the base of the brain sometimes, and base of the brain injury results in disinhibition and impulsivity, and that's a well-known effect of injury to that part of the brain.

I don't know how many people know Phineus Gage, who was railroad worker in Vermont and got a spike through his frontal lobe and lived as a wastrel thereafter. Even though he may have had the same IQ points, he was, you know, a disinhibited -- whatever he was, in all the New York Times articles on the brain.

Anyway --

Q Is it your opinion that the injuries that Estella suffered, those diffuse injuries to the brain, including the seizures and hemorrhages in the thalamus and various other parts of the brain, are those the kinds of injuries that can cause the kinds of behaviors that are noted and the cognitive deficits?

A Yes, I think they are. I know they are.

Q One more thing.

In terms of the size, there is again a contention that because the size of the hemorrhage is very, very small, that that would preclude any kind of causal relationship.

What is your opinion?

A Well, I mean, if you have a stroke in an adult, for

example, well one blood vessel closes over and then there is injury to that part the brain, it's more predictable about what the effects are going to be.

A child's brain is plastic, which is good and bad, but the point is in this instance it's not so much that one dot, as I see it, as the encephalopathy that was described from the beginning and that we know would have taken place on the basis of the metabolic arrangements that occur with hypernatremia and with seizures.

So I think that's interesting, and you can -- there's a lot of speculation. Right hemisphere is more involved with attention than left hemisphere, and there's a lot of this neurologizing, some of which I believe in and some of which I think is overplayed.

I think you have an injury to the brain, diffusely you're going to have some aftereffects along these lines.

That's kind of simplistic, but I think probably more complex than it appears.

Q Is it likely that these kinds of deficits manifest themselves over time in different ways?

A Yes.

Q And how so?

A Well, I already mentioned that the hyperactivity dies down in everyone, what adults describe as inner restlessness or impulsive decision making.

There's a phrase: Ready, shoot, aim, that you use to sort of describe some of the kids as they get to be teenagers. See a car; take a car, whatever. I'm not going to get into great hyperbole about that.

So the point is it manifests differently, and it can be subjective feelings, inability to, you know, hold on to tasks or relationships.

So there's different -- and that merges into personality development from -- that may have a variety of antecedents. So it's not that easy to tease out relative contributions. I mean, there is a similarity that's noted in children with this label as they grow up that many of them have behaviors that impair their functioning to varying degrees and to varying extents.

Q Do you have an opinion as to whether or not it is more likely than not that Estella will continue to have these kinds of impairments?

MR. GIEDT: I'm going to object, your Honor. This was nowhere in his report and just wasn't part of his analysis.

THE COURT: I would still be interested in the opinion.

MR. GIEDT: Thank you.

A What I do say in the report is that I have concerns that what I notice now will have some implication as the tasks

for inferential reasoning and problem solving and organizational skills become more complex. So I had said that and I believe that. That is my concern, because more likely than not, there will be some of these effects over time.

MR. APPEL: I don't have any further questions for you right now.

THE COURT: Doctor, did you want to take a break before we go to cross-examination?

THE WITNESS: I'm fine.

THE COURT: You're fine?

MR. GIEDT: Can we take a break, your Honor?

THE COURT: Why don't we take five minutes for a break and we'll take the regular break after the cross-examination.

THE CLERK: All rise.

Court is in recess.

(Recess.)

THE CLERK: All rise for the Honorable Court.

Court is open. You may be seated.

THE COURT: Mr. Giedt.

MR. GIEDT: Thank you, your Honor.

CROSS-EXAMINATION

BY MR. GIEDT

Q Good morning, Dr. Hart. My name is Anton Giedt. I

represent the United States in this matter.

A Good morning.

Q Dr. Hart, when you're evaluating brain injury and any potential neurological sequelae associated with it, is it fair to say that one of the fundamental principles in neurology is something called, the term of art, localize the lesion?

A Is it fair to say -- what did you say?

Q That is one of the fundamental principles when you're evaluating brain injury and potential neurological sequelae.

A Sure, yes.

Q And would you also agree that the two building blocks of that term of art "localize the lesion" are the location of the insult and determining its size?

A Depending on what the situation is. We know now that there are networks -- in fact, a lot of the information over the last 10 years, 15 years, is talking about neuro networks. And interruption anywhere along these networks can give rise to various deficits. So in the crude sense, this is before the era of CT and MRI, you were looking at any paresis, you wanted to know is it on the left side, the front or the back? But we are talking about behavior, and there's circuitry in networks that are accepted now even to be more significant than in a single lesion.

Q But back to the gross sense, just in layman's terms, if

Q And that can occur due to several different causes, birth hypoxia, infections of the mastoid, sinuses and the ear bleeding, bleeding disorders, connective tissue disorders; is that true?

A It sounds like it's from a textbook. So I assume it's true.

Q And in Estella's case, the CVT, I think you testified on direct, was due to hypernatremic dehydration?

A I believe it was, yes.

Q Turning to ADHD now.

A Yes.

Q Is it fair to say that when you're evaluating a child for possible causes of inattention you would consider, at least, major life stressors?

A You know, that's pretty well been discounted in the literature. Life stressors can affect behavior. If you think specifically inattentiveness, it depends on if it's persistent and the time line, whether it's episodic. I mean, there are many factors that go into evaluating any symptom.

So life stressors causing inattentiveness as such, I suppose if you're preoccupied you might have a period when to the outside world you look inattentive if you're worried about something happening at home, but that would be an episodic kind of thing rather than a persistent finding.

Q My question is, in evaluating a child for possible causes of inattention, would you consider major life stressors?

A In evaluating a child for anything, whether it's seizures or headaches or anything else, I try to take a complete history and look at all factors that might be affecting the child's functioning, yes.

Q And is it fair to say that those life stressors could include a long-distance move, absence of a parent for a significant length of time?

A Are life stressors --

Q Could it include --

A Do life stressors include the above --

Q Yes.

A -- or does inattentiveness result from those causes?

I'm not sure I understand your question.

Q Do life stressors include -- could they include a long-distance move?

A Yes, life stressors could include a long-distance move.

Q The absence of a parent for a significant length or time?

A Or the absence of a parent for a significant length of time, yes.

Q Birth of a newborn child or sibling?

A These are all life stressors or events in a family's

A I don't -- there is actually no long-term follow-up studies of hypernatremia in infants. That's an area of potential research, I suppose, if they could get enough cases.

Vascular injuries, it's known that any injury to the brain can result in some of these. I've seen that. I have an article on aphasia, some of which were caused by strokes, school problems, inattentiveness. Yes, I think those are generally -- specifically from hypernatremia at three weeks of age, there is no long-term follow-ups that I know of.

MR. GIEDT: Could I have a moment, your Honor?

THE COURT: Yes.

(Counsel conferred.)

MR. GIEDT: No further questions at this time.

THE COURT: Mr. Appel.

MR. APPEL: I have a couple of more, your Honor.

REDIRECT EXAMINATION

BY MR. APPEL

Q Dr. Hart, did you look at other potential etiologies for Estella's cognitive and behavioral problems?

A Yes, I did.

Q And what kinds of things were you looking for?

A Well, I specifically asked the parents about any

history, since we know genetics can play a role in this. What was their history in terms of attention and behavior and education and so on. There was nothing that emerged from my discussion with them that might imply that this was a genetic etiology.

My examination of Estella did not reveal any dysmorphic features, abnormalities of palmar creases or any of the clues that might indicate chromosomal abnormalities. And I noted that Dr. du Plessis, even in the midst of that initial crisis, noted the same, the implication I took meaning just that, that he was looking for other causes.

There was no history of intoxication. I mean she's only a week old. So -- there were no other causes that came to my mind that I could eliminate by looking at her or going over the history. The ones I considered weren't there.

Q So in terms of her behavioral problems and cognitive problems today, did you look at the potential role of psychosocial factors?

A Yes.

I mean, my own belief is that she's more vulnerable and that whatever stressors came her way, namely what's been enumerated, the move of the father, or the impending deployments, or the birth of the new baby, then maybe she was a more vulnerable child to begin with and might be expected to perhaps react -- it's sheer speculation, yes,

but children who are vulnerable on a biological basis weather stressors perhaps less well than others who are sturdier built, for whatever that's worth, yes.

Q What is the general scientific consensus today with respect to the role of those kinds of factors in -- causative role in ADHD and these cognitive problems that we're talking about?

A I think there's general agreement in the literature that this is a neurobiological disorder that is not caused by family stressors, depending on -- you know, as it was just mentioned, if you have a child who is doing well and then suddenly starts crawling under the desk or not paying attention, you wonder if something is going on at home.

But that's not the case here. This is -- what I read and talked with the family about was a persistent pervasive developmental difference from 20 months of age on without necessarily -- maybe with some ups and downs, but not suddenly on a substrate of a normal child, that there suddenly is a period of inattentiveness that persists.

So I'm certainly aware of those factors in any family that's in the armed forces, and I actually did a Well Baby clinic at Hanscom Air Force Base myself in 1965. So I'm familiar with the stressors that military families can be under, but I don't think that this is enough to explain this child's dysfunction.